Falsifying the Link Between P Acne Bacteria and Dermal Cysts Associated with Acne

30 March 2025
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Introduction

It has long been held by dermatologists that acne; the medical condition typified by cysts concentrated in the facial area; is caused by a type of bacteria called P. Acne bacteria. The basic assumption which has been accepted is that greater quantities of this bacteria result in an autoimmune reaction leading to the dermatological symptoms associated with the condition.

Abstract

The only regard in which that assumption is correct is that the condition is autoimmune. Rather than treating facial acne as an autoimmune condition, it is treated as one would treat a bacterial infection. This approach ignores the autoimmune nature of the condition and ignores the fact that P. Acne bacteria is ubiquitous and can be found on anyone's skin. Profoundly, not everyone who has P. Acne bacteria in their dermis experiences clinical acne.

There is a correlation between facial acne and oily skin, but this link is not absolute. There are cases of facial acne which present without oily skin, but this is more rare. Furthermore, it is possible for a person to have oily skin in the absence of excessive numbers of sebaceous filaments. I propose that the necessary ingredient for clinical acne to manifest is not oily skin, but the presences of pores blocked by sebaceous filaments.

I propose that the cysts associated with facial acne as the byproduct of an autoimmune reaction triggered by the agglomeration of chemical signals in clogged pores whereas the pores are clogged, generally, by sebum. Whereas ordinarily, mRNA signals meant to prompt glands to take particular actions sc. to start or stop the production of a chemical would osmote through the skin through open pores, when these pores become blocked, the signals can accumulate to undesirable densities. These messenger molecules, at a sufficiently high density, alter the apparent mRNA profile or "scent," if you will, of surrounding cells, causing the immune system to falsely identify the cells as being associated with an infection. In cases of facial acne, the immune system is not reacting to the presence of P. Acne bacteria. Correlative studies have found that P. Acne bacteria can be found in greater quantities on the surface of the skin of those afflicted with acne, most likely, because the oils capture this bacteria and allow it to float to the surface of the skin where it can be sampled more readily whereas in a patient with normal or dry skin, the bacteria resides deep within pores where it cannot readily be collected by swabbing.

Medications such as isotretinoin were highly effective (prior to their recall) in remedying the condition because it had the effect of permanently crippling or

disabling the sebaceous glands, resolving the problem of the inappropriate accumulation of the mRNA signals.

Facial acne most typically resolves itself with age as hormones such as testosterone exacerbate autoimmune reactions, particularly those of an inappropriate nature such as in the case of the inappropriate targeting of healthy tissues by the immune system. As serum levels of this hormone decline with age, it becomes increasingly unlikely that an individual with or without clinically diagnosed acne would experience acne-like cysts in any context. In nearly all documented cases in which acne does not resolve with age, the patient has other underlying autoimmune disorders which would explain the inappropriate targeting of the dermis by the immune system.

Conclusion

Efforts to treat this condition should be focused upon shutting down the sebaceous glands primarily and, only if this fails, addressing the inappropriate autoimmune component, given the risk of undesired side-effects associated with immunomodulator drugs.